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Lyvers, Michael; Thorberg, Fred Arne; Ellul, Adrienne; Turner, Jan; Bahr, Mark

Published in:
Personality and Individual Differences

DOI:
10.1016/j.paid.2009.10.030

Published: 01/02/2010

Document Version:
Peer reviewed version

Negative Mood Regulation Expectancies, Frontal Lobe Related Behaviors and Alcohol Use

Michael Lyvers, Ph.D. [a]

Fred Arne Thorberg, M.A. [bc]

Adrienne Ellul, Postgraduate Diploma of Psychology

Jan Turner, Postgraduate Diploma of Psychology

Mark Bahr, Ph.D. [a]

[a] Department of Psychology

Bond University

Gold Coast Qld 4229 Australia

Corresponding Author: Michael Lyvers

Phone: (61) (75) 5952565

Fax: (61) (75) 5952540

Email: mlyvers@bond.edu.au

[b] Institute of Health and Biomedical Innovation

[c] School of Psychology and Counselling

Queensland University of Technology, Qld 4059, Australia

Abstract

Negative mood regulation (NMR) expectancies have been linked to substance problems in previous research, but the neurobiological correlates of NMR are unknown. In the present study, NMR was examined in relation to self-report indices of frontal lobe functioning, mood and alcohol use in 166 volunteers of both genders who ranged in age from 17 to 43 years. Contrary to expectations based on previous findings in addicts and problem drinkers, scores on the NMR Scale did not differ between Low Risk and High Risk drinkers as defined by the Alcohol Use Disorders Identification Test (AUDIT). However, NMR scores were significantly negatively correlated with all three indices of frontal lobe dysfunction on the Frontal Systems Behavior Scale (FrSBe) Self Rating Form as well as with all three indices of negative mood on the Depression Anxiety Stress Scales (DASS), which in turn were all positively correlated with FrSBe. Path analyses indicated that NMR partially mediated the direct effects of frontal lobe dysfunction (as indexed by FrSBe) on DASS Stress and DASS Depression. Further, the High Risk drinkers scored significantly higher on the Disinhibition and Executive Dysfunction indices of the FrSBe than did Low Risk drinkers. Results are consistent with the notion that NMR is a frontal lobe function.

Keywords: negative mood regulation expectancies, alcohol, frontal lobe

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1. Introduction

Catanzaro and Mearns (1990) defined the concept of generalized expectancies for negative mood regulation (NMR) as beliefs concerning one's ability to terminate or alleviate negative mood states through one's own efforts. Such beliefs vary across individuals, appear to be relatively stable traits, are negatively related to anxiety and depression and are positively related to active coping strategies (Drwal, 2008; Lyvers, Thorberg, Dobie, Huang & Reginald, 2008; Kassel, Bornovalova & Mehta, 2006: Mearns, 1991).. As people with substance disorders frequently cite a need to alleviate negative mood states as a major motive for their alcohol or drug use (Cooper, Frone, Russell & Mudar, 1995; Wills & Shiffman, 1985; Woody, Urschel & Alterman, 1992), Thorberg and Lyvers (2006) examined mood as well as NMR expectancies in 158 volunteers, including 99 clients enrolled in addiction treatment programs who had been abstinent from drugs and alcohol for at least two weeks (and thus were well past acute withdrawal). Addicts reported significantly higher levels of negative moods (stress, anxiety, depression, as assessed by the Depression Anxiety Stress Scales, or DASS; Lovibond & Lovibond, 2002), more intense moods (as assessed by the Affect Intensity Measure, or AIM; Larsen, 1984), and lower NMR expectancies (assessed by the NMR scale; Catanzaro & Mearns) compared to non-addict controls. The findings were consistent with the notion that inherently poor NMR promotes substance abuse as a means of coping with affective distress (Catanzaro & Laurent, 2004; Kassel, Jackson & Unrod, 2000). However, given recent evidence that addiction can induce persistent negative mood states such as depression and anhedonia due to chronic drug-induced alterations of brain dopamine systems (Goldstein & Volkow, 2002; Koob & LeMoal, 2001), an alternative interpretation is that the emotional dysregulation reported by addicts in Thorberg and Lyvers' study was a result, rather than predisposing cause, of their substance disorder. In the present study, alcohol consumption and NMR expectancies were examined in a non-clinical sample in order to determine if heavier and riskier alcohol use would be associated with lower NMR in social drinkers. If confirmed, such a relationship would lend support to the idea that inherently low NMR is a trait that predisposes to substance abuse.

Although the neurobiological underpinnings of NMR expectancies are unknown, the frontal lobes appear to play a major role in mood regulation. For example, the prefrontal cortex is directly involved in the regulation of negative emotions via inhibition of limbic areas such as the amygdala, as revealed by functional neuroimaging (Phan et al., 2005). The prefrontal cortex is also often reported to be dysfunctional in addicts according to both neuropsychological and brain imaging measures (Giancola, 2000; Goldstein & Volkow, 2002; Lyvers, 2000). Brain maturation in adolescence includes integration between prefrontal and limbic regions involved in both executive functioning and the inhibitory regulation of emotional behavior (Benes, Turtle, Khan, & Farol, 1994; Paus, 2005; Steinberg, 2005). Deficits in executive cognitive functioning and affective self-regulation associated with prefrontal dysfunction may thus predispose to substance problems, with relatively impaired prefrontal inhibition and control mechanisms predating the onset of problematic alcohol or other drug use (Agrawal & Lynskey, 2008; Lyvers, Czerczyk, Follent & Lodge, in press; Oscar-Berman & Marinkovic, 2007). The present study examined NMR expectancies in relation to both risky alcohol consumption and indices of prefrontal cortical functioning using self-report measures. We expected to find risky drinking associated with lower levels of NMR in a non-clinical sample, consistent with the hypothesis that inherently low NMR predisposes to heavier or more problematic substance use (Thorberg & Lyvers, 2006). We also expected lower NMR to be associated with higher scores on all three frontal lobe dysfunction indices of the Frontal Systems Behavior Scale (FrSBe; Grace & Malloy, 2001) Self-Rating Form, reflecting the presumed role of the frontal lobes in NMR. Negative moods as assessed by the Depression Anxiety Stress Scales (DASS) were also expected to be negatively related to NMR in line with previous work and consistent with theory (Lyvers et al., 2008; Thorberg & Lyvers, 2006). Finally, the Disinhibition and Executive Dysfunction measures of the FrSBe were expected to differentiate high risk from low risk drinkers as found in a recent study by Lyvers et al. (in press).

2. Method

2.1. Participants

The 166 participants (59 males, 98 females, 9 unspecified) included 93 undergraduate students recruited from Bond University who received 1 credit point towards their assessment in introductory psychology classes. The remaining 73 participants were recruited from local shopping centers and were offered no incentives. Ages ranged 17 to 43 years (M = 24.14 years, SD = 6.49). Student and community samples did not differ in age or gender composition. However, as anticipated the student sample reported significantly more years of education than the community sample, $t(153) = 4.536$, $p < .0001$. Further, though all participants were at least occasional consumers of alcoholic beverages, the community sample contained a higher percentage of risky drinkers (as defined by score on the Alcohol Use Disorders Identification Test of 8 or higher; 58%) than the undergraduate student sample did (41%), $X^2(1) = 4.55$, $p = .04$.

2.2. Materials

The *Negative Mood Regulation (NMR) Scale* is a 30-item questionnaire developed by Catanzaro and Mearns (1990) to measure generalized expectancies to alleviate negative moods. Participants are asked to indicate the degree to which they believe their use of various coping strategies can counteract a negative mood state. Each item is scored on a five point Likert scale ranging from "Strongly disagree" to "Strongly agree" with a statement completing the stem, "When I'm upset I believe that....". Examples of items include "I can do something to feel better," "planning how I deal with things will help," and "wallowing in it is all I can do." Each item is scored on a five point Likert scale ranging from "Strongly agree" to "Strongly disagree." A high score indicates a strong belief that one can alleviate one's own negative moods through non-pharmacological means. Factor analysis has shown that the NMR scale is unidimensional (Catanzaro & Mearns, 1990) and correlates in theoretically predicted ways with instruments assessing anxiety, depression, emotional states and coping responses (Drwal, 2008; Lyvers et al., 2008; Thorberg & Lyvers, 2006). Discriminant validity from social desirability, depression and locus of control has been demonstrated (Catanzaro,1994; Catanzaro & Mearns, 1990; Mearns 1991).

The *Depression Anxiety Stress Scales (DASS;* Lovibond & Lovibond, 2002) is a self-report questionnaire listing negative emotional symptoms and is divided into three subscales measuring

depression, anxiety and stress. Participants rate the extent to which they have experienced each symptom over the past week on a four point Likert scale ranging from "Did not apply to me at all" to "Applied to me very much, or most of the time." The DASS comes in a long form consisting of 42 items and a short form consisting of 21 items. As the 21-item version has several advantages over the longer version in terms of fewer items, a cleaner factor structure and smaller inter-factor correlations (Antony, Bieling, Cox, Enns & Swinson, 1998), it was used in the present study. Items include "I couldn't seem to experience any positive feeling at all" (Depression), "I was worried about situations where I might panic and make a fool of myself" (Anxiety), and "I found it hard to wind down" (Stress).

The Alcohol Use Disorders Identification Test (*AUDIT*; Babor, de la Fuente, Saunders & Grant, 1992) is composed of 10 questions, including 3 quantity/frequency questions (e.g., "How often do you have a drink containing alcohol?"), 3 dependence-related items (e.g., "How often during the last year have you failed to do what was normally expected of you because of drinking?"), and 4 items assessing alcohol-related consequences or harm (e.g., "Have you or someone else been injured because of your drinking?"). AUDIT questions are scored from 0 to 4, with an overall score ranging from 0-40. The suggested cut-off differentiating Low Risk from Hazardous drinking is a total AUDIT score of 8 or higher (Babor et al.). Factor analysis supports construct validity of the AUDIT (Shields, Guttmannova, & Caruso, 2004). Internal consistency (Cronbach's alpha) ranges from .80 (Kane, Loxton, Staiger, & Dawe, 2004) to .94 (Pal, Jena, & Yadav, 2004) and test-retest reliabilities range from r =.87 over one week (Rubin et al., 2006) to r =.93-.95 over four weeks (Bergman & Källmén, 2002; Dybek et al., 2006). An assessment of convergent validity of total and factor scores against the Michigan Alcoholism Screening Test established correlations ranging as high as .97 (Pal et al., 2004).

The *Frontal Systems Behavior Scale* (*FrSBe*) *Self-Rating Form* is a 46-item questionnaire assessing behavioral evidence of dysfunction in three major prefrontal-subcortical systems of the brain (Grace & Malloy, 2001). There are three corresponding subscales: Apathy (poor initiation, reduced drive and interest, 14 items; sample item: "I sit around doing nothing"), designed to assess anterior cingulate dysfunction; Disinhibition (distractibility, problems with inhibition, socially inappropriate behaviour, 15 items; sample item: "I do things impulsively"), designed to assess orbitofrontal dysfunction; and Executive Dysfunction (difficulties with planning, sequencing, working memory, and mental flexibility, 17 items;

sample item: “I repeat certain actions or get stuck on certain ideas”). The standard version of the Self Rating Form of the FrSBe aims to measure behavioral change by obtaining pre-and post-lesion ratings. For the purposes of this study and in keeping with previous research (Lyvers et al., in press; Spinella, 2003; Verdejo-García, Rivas-Péreza, López-Torrecillasa, & Pérez-García, 2006) only overall scores in present time were obtained. Items are rated on a 5 point Likert type scale. The first 32 items represent deficits and are rated accordingly, with the final 14 positively stated items reverse scored. The magnitude of the score on each subscale indicates the degree of impairment. Factor analyses of the FrSBe in several neurological populations have supported the construct validity of the subscales (Stout, Ready, Grace, Malloy, & Paulsen, 2003) with the three factor solution accounting for 40.7% of the common variance. Evidence also supports reliability (Velligan, 2002) with internal consistency ranging from .88 to .91 and three month test-retest reliability of .78. Diagnostic validity has been confirmed for detecting graduated degrees of symptoms of frontal lobe functioning in various clinical samples (Chiaravalloti & DeLuca, 2003; Velligan, 2002), in substance use and abuse populations (Spinella, 2003) and in healthy individuals (Spinella, 2007).

2.3. Procedure

Bond university students were recruited via a notice board on campus for the incentive of 1 credit point toward introductory psychology classes. All students who agreed to participate (n = 98) made appointments to be tested individually in a room on campus. Prior to completion of the survey, the researcher read aloud the explanatory statement attached to the top of each questionnaire packet, informing the student that all information collected would be anonymous with no identifying details recorded. The students were asked to complete all items as honestly and correctly as possible.

For the local community sample (n = 75), people were randomly approached outside local shopping centers on four week days and asked if they would like to take part in the study. An explanatory statement outlining the brief purpose of the study was read aloud to each potential participant. This statement was also attached to the top of each questionnaire packet. Participants were informed that participation was entirely voluntary and that all participants would remain anonymous as no identifying details would be recorded. Those who agreed to take part in the study

were asked to complete all questions as honestly and correctly as possible. They then completed the questionnaires at nearby seating areas. No incentives were given for the community participants. All participants handed back the completed surveys individually to the researcher in a sealed envelope.

3. Results

Intercorrelations among all variables are shown in Table 1. As predicted, NMR was moderately negatively correlated with all three indices of frontal lobe dysfunction on the FrSBe as well as with the DASS scales, and all FrSBe and DASS scales were positively correlated with each other. However, NMR was unrelated to AUDIT, contrary to predictions. AUDIT was moderately positively correlated with Disinhibition and Executive Dysfunction scores on the FrSBe, replicating previous work (Lyvers et al., in press).

A between groups Multivariate Analysis of Covariance (MANCOVA) was conducted, with the independent variables of gender and AUDIT group – the latter defined by the AUDIT cutoff distinguishing Low Risk (AUDIT score < 8) from Hazardous (AUDIT score 8+) drinkers (Low Risk, n = 77; Hazardous or higher = "High Risk," n = 77), yielding identical group sizes (note that 12 participants were excluded from this analysis due to missing gender or age data). AUDIT groups did not differ in age, $t(152) = 1.13$, $p = .26$; however, females comprised 73% of the Low Risk group compared to 52% of the High Risk group, a significant gender difference, $X^2(1) = 8.20$, $p = .004$. Because age was significantly correlated with some variables of interest (see Table 1), it was a covariate. The dependent variables were DASS, NMR and FrSBe scores. According to Pillai's Trace, there was a significant multivariate effect of AUDIT group on the combined dependent variables, $F(7, 143) = 3.25$, $p = .003$. Neither gender nor the interaction approached significance. The univariate effect of AUDIT group was significant for FrSBe Disinhibition, $F(1, 149) = 13.86$, $p < .0001$, and FrSBe Executive Dysfunction, $F(1, 149) = 4.91$, $p = .028$. High Risk drinkers scored significantly higher (suggesting greater orbitofrontal dysfunction) on Disinhibition ($M = 33.88$, $SD = 6.83$) than did Low Risk drinkers ($M = 29.51$, $SD = 7.19$). High Risk drinkers also scored significantly higher (suggesting greater dorsolateral prefrontal dysfunction) on Executive

Dysfunction (*M* = 38.97, *SD* = 7.69) than did Low Risk drinkers (*M* = 35.74, *SD* = 8.64). No other AUDIT group differences approached significance, including NMR. Even when the 24 participants defined by AUDIT as drinking at a Harmful level (AUDIT score of 16+) were compared to Low Risk drinkers, there was no difference on NMR, $t(108) = .434$, *n.s.*.

Finally, to explore the potential mediating role of NMR in the relationships between frontal lobe dysfunction as indicated by FrSBe scores and DASS Depression, Anxiety and Stress scores, a series of three mediational analyses were conducted using EQS (Bentler, 1989). Three separate analyses were conducted to circumvent multicollinearity, as an initial attempt at a single mediational model which incorporated Depression, Anxiety and Stress (DASS) domains failed to converge, and produced runaway path coefficients. The runaway coefficients appeared to result from a degree of multicollinearity between terms and correlated error terms. Running the analysis as three separate solutions allowed convergence of the model, with little loss of information. The initial model also included AUDIT score as an outcome variable, however, this variable was dropped due to near zero correlation between AUDIT and the primary predictor. Figures 1, 2 and 3 show the models for DASS Anxiety, Stress and Depression, respectively. The separate analyses converged with adequate fit (see Table 2). It should however be noted that the NNFI indices indicated a degree of misfit remaining between model and data. The RMSEA was also higher than desirable although standardised RMR for each analysis was acceptably lower than .05 for each solution. This pattern of results may be in part explained by the relatively small *n* available for analysis, however confidence in the solution is enhanced by rapid convergence to a solution in each case.

Table 3 indicates that NMR partially mediated the direct effects of frontal lobe dysfunction (as indexed by FrSBe) on DASS Stress and on DASS Depression. The relationship between frontal lobe dysfunction and DASS Anxiety was markedly different from the Depression and Stress findings. The path coefficients for this model suggest that the direct path between FrSBe and Anxiety was not mediated by NMR. There is evidence for a slight negative relationship between NMR and Anxiety (as shown in Figure 1), however this path is not significant. As such this model fails step 2 of Baron and Kenny's(1986) mediation process.

4. Discussion

Thorberg and Lyvers (2006) found that clients undergoing inpatient treatment for addictions (including alcoholism) reported significantly higher levels of stress, anxiety, and depression on the DASS and lower levels of NMR compared to non-addict controls. In the present study, there were no such differences between those defined as High Risk drinkers and those defined as Low Risk drinkers based on their AUDIT scores in a non-clinical sample of social drinkers that included university students and members of the local community. This was unexpected given the report of Kassel et al. (2000) that alcohol-related problems (as assessed by a modified version of the Hawaii Alcohol Survey) were associated with low NMR in a sample of university undergraduates. In their study, self-reported alcohol problems but not consumption levels were related to NMR, whereas the present study only examined NMR in relation to risky drinking as defined by AUDIT scores. Perhaps only more serious alcohol problems or alcohol-related concerns are related to low NMR. However, in the present study there were 24 participants who were classed by AUDIT as drinking at a Harmful level, and even that extreme group did not differ from Low Risk drinkers on NMR. Differences between the two studies in the outcome measure of drinking may account for the discrepant findings, and/or differences in the samples (e.g., 17 of the 24 drinkers classed as Harmful drinkers by AUDIT were from the community sample rather than the undergraduate student sample in the present study, whereas Kassel et al. examined only university undergraduates).

In any case, the present findings do not support the hypothesis that inherently low NMR predisposes to riskier substance use. The report of Thorberg and Lyvers (2006) of significantly lower NMR and higher DASS scores in alcoholics and drug addicts compared to controls may thus have reflected sequelae of chronic addiction rather than predisposing traits. Although the addicts tested by Thorberg and Lyvers were more than two weeks abstinent from alcohol and drugs, anhedonia and other mood problems in addicts have been reported to persist well past the acute withdrawal stage and are attributed to enduring drug-induced changes in dopamine pathways, including the mesocortical dopamine system innervating the frontal lobes (Goldstein & Volkow, 2002).

The present study found that High Risk drinkers scored significantly higher on the Disinhibition and Executive Dysfunction indices of the FrSBe than Low Risk drinkers, consistent with previous work (Lyvers et al., in press). Further, as expected, NMR was moderately negatively correlated with all three indices of frontal lobe dysfunction on the FrSBe, consistent with the notion that low levels of NMR may reflect poor prefrontal cortical inhibition of limbic areas such as the amygdala involved in generating negative emotions. Also consistent with this idea, the DASS scales were all positively correlated with the FrSBe scales and negatively correlated with NMR. Path analyses indicated partial mediation of the relationships between FrSBe and DASS Depression and Stress by NMR, though this did not appear to be the case for Anxiety. Overall these relationships fit with a variety of neurological evidence which, going back to the famous case of Phineas Gage, identifies mood regulation as a frontal lobe function. However, as the present findings supported the hypotheses that inherent mild dysfunction of orbitofrontal and dorsolateral prefrontal cortex is associated with riskier alcohol consumption (Lyvers et al., in press), and that NMR is related to frontal lobe functioning, the absence of any relationship between NMR and alcohol consumption was perhaps surprising. Further investigation of NMR in the context of alcohol or other drug use, abuse and addiction thus appears warranted.

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Table(s)

Table 1

Intercorrelations among Years of Education (Educ), Age, DASS Depression (Dep) Anxiety (Anx) and Stress, NMR, AUDIT (AUD), and FrSBe Apathy (Apath), Disinhibition (Disin), and Executive Dysfunction (Exec).

	Age	Educ	Dep	Anx	Stress	NMR	AUD	Apath	Disin
Educ	.105								
Dep	-.151	-.081							
Anx	-.221**	-.198*	.675**						
Stress	-.071	-.075	.607**	.694**					
NMR	.009	.172*	-.440**	-.331**	-.368**				
AUD	-.131	-.024	.016	.037	-.060	.030			
Apath	-.108	.071	.440**	.279**	.241**	-.465**	.069		
Disin	-.191**	.073	.291**	.363**	.284**	-.304**	.352**	.492**	
Exec	-.096	.012	.479**	.413**	.347**	-.466**	.264**	.638**	.690**

** $p < .01$ * $p < .05$

Table 2: Mediational model fit for three mediation models individually examining NMR's role as a mediator of anxiety, stress, and depression.

Outcome	Chi	NFI	NNFI	CFI	GFI	RMSEA
Depression	13.30 (2)	.95	.78	.96	.97	.19
Stress	10.06(2)	.96	.83	.97	.98	.16
Anxiety	10.52 (1)	.96	.59	.96	.97	.25

Table 3: Path coefficients for Mediational models individually examining NMR's role as a mediator of anxiety, stress, and depression.

Outcome	Path a	Path b	Path c
Depression	-.50*	-.42*	.68*
Stress	-.50*	-.51*	.64*
Anxiety	-.50*	-.13	.35*

Figure 1: Does Negative Mood Regulation Mediate the influence of executive function on DASS Anxiety?

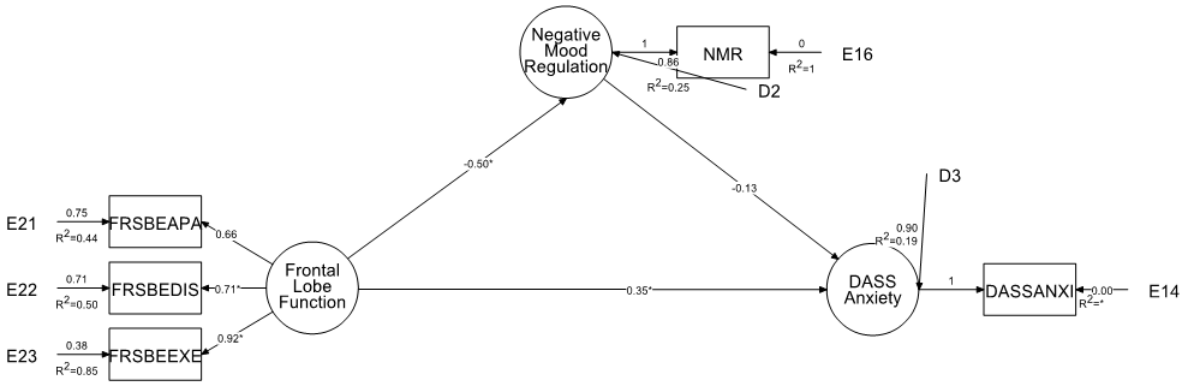

Figure 2: Does Negative Mood Regulation Mediate the influence of executive function on DASS Stress?

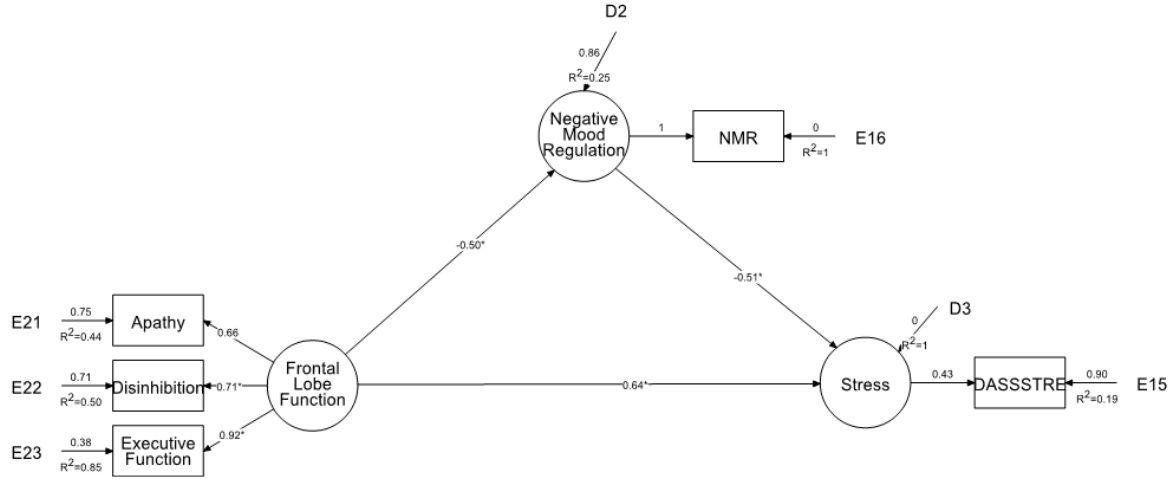

Figure 3: Does Negative Mood Regulation Mediate the influence of executive function on DASS Depression?